# Ferruginol

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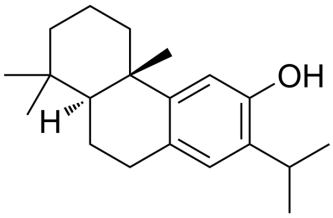
| **Cat. No.:** | HY-N6033 |
| **CAS No.:** | 514-62-5 |
| **Molecular Formula:** | $C_{20}H_{30}O$ |
| **Molecular Weight:** | 286.45 |
| **Target:** | HSV; Apoptosis; EBV |
| **Pathway:** | Anti-infection; Apoptosis |
| **Storage:** | Please store the product under the recommended conditions in the Certificate of Analysis. |




## BIOLOGICAL ACTIVITY

**Description**

Ferruginol ((+)-Ferruginol), a natural diterpenoid, is an inhibitor of the activation of Epstein-Barr virus early antigen (EBV-EA). Ferruginol inhibits the growth of thyroid cancer cells through the induction of mitochondrial apoptosis. Ferruginol has antitumor, cardioprotective, antioxidant, gastroprotective, and neuroprotective activities[1][2][3].

**In Vitro**

Ferruginol (0-160 μM; 24 hours) exerts potent antiproliferative action against thyroid cancer cells, and an $IC_{50}$ of 12 μM for the MDA-T32 cell line. The toxic effects of Ferruginol are less pronounced against normal cells[1].
Ferruginol (0-24 μM; 24 hours) induces apoptotic cell death of MDA-T32 cells. Ferruginol increases Bax expression and decreases Bcl-2 expression dose-dependently[1].
Ferruginol (0-24 μM; 24 hours) inhibits the MAPK and PI3K/AKT signaling pathway of MDA-T32 cells[1].
Ferruginol (0-24 μM; 24 hours) also causes ROS mediated alterations in the MMP of MDA-T32 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

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| Cell Line: | MDA-T32 cells |
| Concentration: | 0-160 μM |
| Incubation Time: | 24 hours |
| Result: | Exerted potent antiproliferative action against thyroid cancer cells. |

Apoptosis Analysis[1]

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| Cell Line: | MDA-T32 cells |
| Concentration: | 0 μM, 6 μM, 12 μM, and 24 μM |
| Incubation Time: | 24 hours |
| Result: | Induced apoptotic cell death of MDA-T32 cells |

Western Blot Analysis[1]

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| Cell Line: | MDA-T32 cells |

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| Concentration: | 0 μM, 6 μM, 12 μM, and 24 μM |
| Incubation Time: | 24 hours |
| Result: | Blocked the MAPK and PI3K/AKT signaling pathway. |

**In Vivo**

Ferruginol (20 mg/kg; p.o.; daily; for 4 weeks) exerts cardioprotection manifested as enhanced cardiac function and reduced structural damage and apoptosis. The transcriptome and other results revealed that Ferruginol facilitates PGC-1α-mediated mitochondrial biogenesis and fatty acid oxidation (MB and FAO) by increasing the expression of PGC-1α and concurrently promoting the expression of SIRT1-enhancing deacetylase SIRT1 deacetylating and activating PGC-1α[3].

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| Animal Model: | Male C57BL/6 mice (20 g, 8-10 weeks old) with Doxorubicin (DOX)-induced cardiotoxicity (DIC)[3]. |
| Dosage: | 20 mg/kg |
| Administration: | Administered intragastrically; daily; for 4 weeks |
| Result: | Relieved Doxorubicin-induced cardiac structural and functional lesion. |

## REFERENCES


[1]. Manabu Iwamoto, et al. Potential antitumor promoting diterpenoids from the stem bark of Thuja standishii. Planta Med. 2003 Jan;69(1):69-72.

[2]. Guoqing Luo, et al. Ferruginol Diterpenoid Selectively Inhibits Human Thyroid Cancer Growth by Inducing Mitochondrial Dependent Apoptosis, Endogenous Reactive Oxygen Species (ROS) Production, Mitochondrial Membrane Potential Loss and Suppression of Mitogen-Activated Protein Kinase (MAPK) and PI3K/AKT Signaling Pathways. Med Sci Monit. 2019 Apr 21;25:2935-2942.

[3]. Weili Li, et al. Ferruginol Restores SIRT1-PGC-1α-Mediated Mitochondrial Biogenesis and Fatty Acid Oxidation for the Treatment of DOX-Induced Cardiotoxicity. Front Pharmacol. 2021 Nov 24;12:773834.





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